

In the specification:

Replace the original paper copy of the sequence listing with the substitute paper copy of the sequence listing filed herewith.

Paragraph beginning at page 7, line 13, has been amended as follows:

Figures 10A and 10B illustrate that the HSE element confers heat-shock, and HSF-1d202-316, inducibility on the melanoma-specific Tyr 300 base pair promoter. Figure 10A is a schematic of a nucleic acid construct according to one embodiment of the invention, where the consensus HSE element (SEQ ID NO:2) is cloned upstream of the Tyr-300 promoter element and is separated from the start of the promoter by a linker (i.e., ACTGGAGAT, SEQ ID NO:7) of either 0 base pair or 10 base pair (i.e., a full turn of the DNA helix) or by 5 base pair (a half turn of the helix). Figure 10B shows the results of a transient transfection assay using the following constructs: Tyr-300-GM-CSF, HSE-Tyr 300-FULL-GM-CSF, TDE-SV40-GM-CSF, HSF-1d202-316 plasmid, and a construct comprising the HSF-1 cDNA.

Paragraph beginning at page 18, line 23, has been amended as follows:

In one embodiment according to the invention, an amplification promoter element, such as a heat shock element is used to amplify the expression of a downstream therapeutic transgene. Heat shock elements ("HSEs") are sequences found within the first 100 base pair 5' of the RNA start site of eucaryotic heat shock genes (see, e.g., Sorger, P.K. Cell 65:363 (1991), the entirety of which is incorporated by reference herein). Heat shock genes, such as Hsp70 genes, from different species differ in the number and orientation of HSEs and in the types of other transcription factor-binding sites found upstream. HSEs include the sequence nGAAn, repeated at least two times in head-to-head or tail-to-tail orientation (nGAAnnTTCn (SEQ ID NO:5) or nTTCnnGAAn (SEQ ID NO:6)), and in one embodiment of the invention, the HSE comprises at least two nGAAn sequences.

Paragraph beginning at page 29, line 9, has been amended as follows:

For the analysis of melanoma specific gene expression, plasmids and viruses were transduced into either melanoma (MeWo, Mel624, A378M, B16 or 1735) or non-melanoma (HT1080; 293; Vero, Tel.CeB6, HeLa, CMT93) cell lines. The human tyrosinase promoter plasmids (300 base pairs; 115 base pairs or 65 base pairs) are as described by Bentley et al., *supra*, and Diaz, et al., *supra*. The TDE-SVO plasmid consists of three repeated copies of the 20 base pairs Tyrosinase Distal Element (TDE) upstream of the minimal SV40 basal promoter (Promega) (Diaz, et al. J. Virol. (1998) 72:789-795, the entirety of which is incorporated by reference herein). The cDNA of the mutated HSF-1 transcription factor is described in Zuo, et al., Mol. Cell Biol. 15:4319-4330, (1995) (the entirety of which is incorporated by reference herein) and consists of a deletion of the wild type HSF-1 cDNA corresponding to amino acid positions 202-316. The HSE element - 5'-AGAATGTTCTAGAAG-3' (SEQ ID NO:2) was synthesized as a consensus sequence which confers heat shock and HSF-1 responsiveness on heterologous genes as described in Amin, et al., Mol. Cell Biol. 8:3761-3769 (1988) and Goldenberg, et al., J. Biol. Chem. 263:19734-19739 (1988), the entireties of which are incorporated by reference herein.

Paragraph beginning at page 36, line 26, has been amended as follows:

Since levels of expression from highly cell type-specific promoters are generally not therapeutic, even when highly potent genes (e.g., cytotoxic) genes such as FMGs are used, in one embodiment, an amplification promoter element was operably linked to an FMG under the control of a highly cell type-specific promoter. In one embodiment, an amplification promoter comprising a consensus HSE sequence (5'-AGAATGTTCTAGAAG-3'; SEQ ID NO:2) modified from the construct described in Goldenberg, et al., *supra*, and Todyry, et al., *supra*, was synthesized.